

length of the apnœic period, which fell to from ten to twelve seconds. Another new point made out by M. Biot was that the peculiar sphygmographic trace of aortic insufficiency was more marked during the apnœa, although the cardiac beats were most numerous at the same time. From this he argues that the arterial tension is less than during the period of dyspnœa. Again, the beats of the heart began to quicken as soon as the breathing became superficial, and to slow immediately on the resumption of breathing after its cessation; so that there was a distinct cardiac rhythm as well as a respiratory one, but alternating in time, the one ascending as the other was descending, and *vice versâ*. This is extremely well shown by the register of the tracings of the heart and chest movements. The patient experienced only a vague feeling of uneasiness during the dyspnœa, whilst his intelligence became slightly clouded during the apnœa. The rhythmical irregularity is obviously so dependent on some interference with either the cardiac or pulmonary innervation, or with both, that the attention of physiologists and physicians was quickly drawn to the pneumogastric nerves. Von Dusch, in 1867, describes this symptom as present in cerebral affections, tumours of the brain, basal meningitis, uræmic coma, etc. We think that in this there must be some mistake. The sighing respiration in late stages of meningitis is so very distinct from that which we have been considering, that we should scarcely think there was any possibility of their being confounded; yet we fear that Von Dusch must have done this, or otherwise it is impossible to explain the variety and number of cases in which he has noticed so very rare and striking a phenomenon. Schweig's explanation, that it is due to a contraction of the posterior lacerated foramen, and so causing compression of the vagi, is too absurd to require refutation. Prof. Laycock attributed the phenomenon to a sentient paresis of the respiratory centre, not necessarily dependent on structural or other disease of the heart; but this must happen more frequently by far than do cases of Cheyne-Stokes' respiration. Dr. Hayden would add to this a want of oxygen in the tissues generally, but the tracings of M. Biot clearly prove that, in some cases at least, the action of the heart, and consequently the rapidity of the arterial circulation, is increased not during the dyspnœa, but during the apnœa. Traube thinks that the accelerated respiration is due to inordinate stimulation of the pneumogastric nerves by the accumulation of carbonic acid in the lungs, and the apnœa to their prolonged over-action; whilst Filehne of Erlangen calls in aid the vaso-motor centre, believing that the activity of the respiratory nerve-centre is comparatively much more lowered. M. Biot's registers undoubtedly favour Traube's theory, but the origin of the peculiar rhythm and its steady persistence for weeks in some cases are still without any adequate explanation. More work is demanded on the part of physicians, pathologists, and physiologists, before a satisfactory hypothesis can be hoped for, and M. Biot shows well one mode in which this must be done.—*Lancet*, Feb. 17, 1877.

14. *Gonorrhœal Endocarditis*.—Dr. MARTY, of the Val-de-Grâce, has a paper on this subject in the *Archives Générales de Médecine* for December. It is based on a case under the care of Professor Poncet, but there is a pretty full *résumé* of a number of cases previously recorded.

D., aged twenty-two, was admitted to the Val-de-Grâce under M. Poncet, August 17, 1876. There were no rheumatic or cardiac antecedents in his family, and he had never suffered from any articular symptom. When five years old he stated that he had been subject to palpitations, but for several years he had been in the habit of lifting heavy weights without any trouble. His gonorrhœa began on August 15, after eight days' incubation. The attack was of moderate severity; rather sharp pain, and abundant discharge; a little local inflammation; no general phenomena. In a few days he had some gastric catarrh, but not the least joint-pain. From August 17 to September 22, the gonorrhœa followed its usual course. On September 22 he had repeated rigors and intense headache. His gonorrhœa diminished without being completely suppressed. On the 26th the first sound at the base was altered, and on the 28th there was a systolic murmur, heard best at the junction of the

third left costal cartilage with the sternum, and conducted along the aorta. His temperature was 101.4° in the morning, and 103.2° in the evening. It had been so within a few points for four days, but the patient did not show much distress. His tongue was moist, his pulse full and strong. He was treated with a blister over the cardiac region, and digitalis. On August 4 there was some vomiting and a little intermittence of pulse. On the 5th there was some presternal pain; the pulse was full, but intermittent. The murmur was traced along the vessels of the neck. The discharge was almost *nil*. On the 6th the evening temperature again rose to 102.9° , and there was palpitation for the first time. There was abundant sweating; constipation; tongue moist. On the 8th there were some râles heard in the lungs, believed to be due to œdema. After the 13th the patient gradually improved. On the 23d there was nothing to note except that he was feeble, that he still sweated, that the murmur was scarcely altered, and that the urethral discharge had quite disappeared. On the 26th his general improvement was still maintained, but the gonorrhœa reappeared. Dr. Marty believed that the murmur was due to the occurrence of proliferating endocarditis at the level of or on one of the aortic valves.

We have only space to summarize Dr. Marty's general conclusions, which are as follows: Gonorrhœa may be complicated with inflammation of all the serous membranes, and may act in a direct way on each of them. Rheumatism is by no means a necessary middle term between the specific lesion and the lesion of the serous membrane, although the cases of coexistence of the two complications are most frequent. The cardiac complications are rare. Of the several orifices the aortic one is most commonly attacked. Endocarditis appears to have occurred as frequently as pericarditis, if not more so.—*Med. Times and Gaz.*, Dec. 23, 1876.

15. *Anomalous Conduction of Mitral Murmurs.*—At a late meeting of the Medical Society of London, Dr. SANSOM related (*Med. Times and Gaz.*, Jan. 27, 1877) two cases recently under his care in the London Hospital, illustrating anomalous conduction of mitral murmurs. The patient in the first case was a man, twenty-four years of age, who had had three attacks of acute rheumatism, and had on a former occasion been a patient in the London Hospital, when a presystolic mitral murmur was noted. There was now a loud musical first-sound murmur, and a systolic thrill could be detected, whose maximum was a little to the left of the base of the ensiform cartilage. It was so strongly marked in the tricuspid area that most competent observers thought it must be due to tricuspid regurgitation. There was now no presystolic murmur. Dilatation of the right side and the usual signs of failing heart preceded death. At the autopsy the mitral orifice was found to be narrowed so as to admit only one finger, and to be surrounded by a calcareous ring. The calcareous matter was attached in largest mass to the interventricular septum. Dr. Sansom considered that this dense calcareous material had acted as a conductor of sound to the interventricular septum, and so by the right apex to the ear; hence the abnormality of position of the murmur. In the second case the patient was a man aged fifty, whose only previous illnesses were chorea at eight years of age, and scarlatina at fourteen. Lately he had had rheumatoid pains, but no fever. On auscultation, a musical presystolic murmur could be heard over an area not greater than a circle of one inch and a half in diameter, just to the left of the base of the ensiform cartilage. A little to the left of this spot there was a loud rough first-sound murmur. Twelve months after first coming under observation the presystolic murmur ceased to be audible. Before death, dilatation of the right side was manifest, with venous pulse and throbbing liver. To relieve ascites, paracentesis abdominis was performed, and it was noted that the urine, which just previously had been highly albuminous, now contained very little albumen, but was highly saccharine. The diagnosis in this case was arrived at by experience of the former. It was that there was mitral stenosis, the characteristic presystolic murmur of which had been conducted by dense material to the interventricular septum, and thence to the apex; that, considering the musical character of the murmur, the valve aperture was probably a calcareous